These sensations which are not evoked by an external object are termed hallucinations, and in persons with diseased judgment are interpreted as realities.

5. All sense organs are subject to corresponding sensations.

6. We find in certain peculiar states of the brain, in that of anæmia which produces normal sleep, in that of physical alteration which paralyzes a certain number of our intellectual faculties, the condition characterized by dreams, somnambulism, and hysteria.

7. Hallucinations can be produced in the healthy man by exciting the imagination, securing the attention, or by persuading imaginary sensations.

8. The very fact that our organs of sense are liable to error should render us very careful in estimating their evidence.

9. Provoked hallucinations are obtained with great facility in persons plunged in pathological sleep in consequence of certain procedures or of a special neurotic state.

10. The pathological sleep is preceded or accompanied by such modifications of innervation as lead to catalepsy, hyper-excitability of nerves and muscles, anæsthesia, hyperæsthesia, etc.

11. It is produced in persons who are predisposed thereto by certain procedures known as magnetic passes, hypnotism, physical impressions, such as music, thunder, loud noise, brilliant light, certain perfumes, etc., or by moral impressions, great fright, disappointment, great joy, etc.

12. The phenomena which accompany the condition should be severely criticized in order to detect those due to deception, etc.

13. In the actual state of our positive consciousness it is not necessary in order to explain the so-called magnetic phenomena, to have resource, with Mesmer and his followers, to the hypothesis of a fluid endowed with marvellous properties.

14. The essential cause of these phenomena does not reside in the magnetizer but in the magnetized and in a neurotic state peculiar to the latter.

15. Positive science cannot deny the possible existence in the universe of entirely unknown forces, but it can only speculate as to those which have been duly demonstrated. Without doubt the extent of the unknown is immense, and we should aspire to new conquests by serious and methodic work, eliminating all error due to a diseased imagination or to new scientific interests.—*Rev. Internat. des Sci. Biol.*, July 15, 1882.

The Movements of the Iris and their Mechanism.

As the results of forty-nine experiments on dogs, cats, goats, and more especially white rabbits, the author, M. Moriggia, comes to the following conclusions, which are recorded in *Moleschott's Unters. zur Naturl.*, etc., xiii., 1, p. 1: In animals which have been kept for several weeks in the dark, the pupil generally dilates more widely in the dark, and contracts more completely in the light, than in those which have been kept in the ordinary conditions as regards light and darkness. Under the same conditions the mydriatics and myotics act much more promptly. The pupil, when dilated (though not to its fullest extent) by atropin, does not contract in response to light. If a concentrated solution of atropin be dropped into one eye, in goats and rabbits, it has little or no effect on the pupil of the other eye; in albino rabbits, however, it acts very powerfully on the other eye. Even when the pupil is dilated as widely as possible this condition yields at once when the superior cervical ganglion of the sympathetic is extirpated. On stimulating the uninjured cervical sympathetic by means of an induced current, the pupil is widely dilated. The mydriatic action of atropin is less marked on the side from which the superior cervical ganglion has been removed, whether its removal has been effected before or after the application of the atropin. On subjecting rabbits to very severe and general muscular exertion, a transient, but very marked, mydriasis was noticed; but this was always less marked on the side on which the uppermost cervical ganglion was wanting. The highest degree of dilatation induced by the use of atropin gives place to contraction when the animal is caused to bleed to death through a wound in the heart. On the other hand, the myosis induced by copious hemorrhage passes into pronounced and permanent mydriasis if atropin be dropped into the eye of an animal just dead, even on that side on which the superior cervical ganglion of the sympathetic had previously been extirpated.—*Glasgow Med. Journal*, May, 1882.

Results of Obstructed Pulmonary Circulation.

M. FRANÇOIS FRANCK has communicated to the Bordeaux Medical Society some instructive results of the experimental obstruction to the pulmonary circulation. The effect of a hindrance to the circulation of blood through the lungs in causing engorgement of the right heart, and secondary insufficiency of the tricuspid orifice, is one of the most familiar facts in medicine. M. Franck's object was to produce this experimentally in order to study more closely the physiological conditions and symptoms, one of which was the chief subject of the communication—viz., the indications of the pulmonary obstruction which are afforded by the jugular veins. The phenomena may be produced by the tracheal insufflation of irritant vapours, such as sulphurous acid, the animal having been rendered insensible by chloral or motionless by curara. This sets the whole contractile mechanism of the lung into energetic action, the muscular tissue of the bronchial tubes and of the vessels. Hence there is a retraction of the whole lung, indicated by the depression of the costal walls, and there is also a diminution of the calibre of the pulmonary vessels, causing an engorgement of the right side of the heart. If the obstruction lasts long enough and is sufficiently intense, the feeble parts of the wall of the right ventricle—*i. e.*, the part above and to the right—yield to the excess of internal pressure, and drag outwards the papillary muscles, and the chordæ tendineæ, and with them the free edge of one of the segments of the tricuspid valve. By this mechanism, which was pointed out by King in 1837, temporary tricuspid regurgitation occurs, and causes a venous reflux, visible as the jugular pulsation synchronous with the ventricle systole. The tricuspid insufficiency and venous pulse continue as long as does the engorgement of the right side of the heart. This "safety-valve action," as King called it, may be thus constantly produced artificially. The analogy between this induced condition, and that which occurs in every paroxysm of spasmodic asthma, is sufficiently close, and there can be no doubt that in the latter the same cardiac phenomena occur. It is probable that the production of this tricuspid reflux is facilitated by certain conditions of the heart itself. The diastolic relaxing influence of the pneumogastric nerve undoubtedly plays an important part in the experimental production of this condition, and no doubt also in its clinical production. M. Potain has insisted on the transient tricuspid regurgitation which seems to be produced by a nervous mechanism under the influence of strong irritation of the abdominal viscera, acute affections of the liver, intestine, and stomach. The reflex action may probably be double, and the effect produced not only by cardiac inhibition, but also by obstruction to the pulmonary circulation.—*Lancet*, July 22, 1882.